are less active than lean children, the published data are equivocal (8-13).

Television viewing was more prevalent among the obese than the nonobese girls, although the difference only approached significance (P=.06). This finding is consistent with the findings of others (8,14,15). Thus, it appears that in Mexican-American children, as well as in white children, television viewing may be an important predictor of obesity.

APPLICATIONS

Childhood obesity in the Mexican-American population is a growing public health problem. Because childhood obesity has been identified as a risk factor for adult obesity and its related health problems, it is imperative to intervene with primary and secondary preventive measures. More information regarding risk factors for childhood obesity in the Mexican-American population is needed before an effective intervention can be developed and implemented. At present, limited descriptive data exists on childhood obesity, especially in Mexican-American families. Additional data are needed to identify the risk factors leading to childhood obesity. ■

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Urinary lactose excretion is not an index of lactation performance

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Noninvasive biomarkers are needed to evaluate a person's nutritional status. Currently, however, a noninvasive biomarker for evaluating lactation performance is not available. The most widely used method requires weighing the infant before and after each feeding for a period of 72 hours (1-4). Be-

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cause this procedure is tedious and time consuming, it is impractical for large-scale population studies or for the assessment of lactation performance in clinical patients.

Strand and Johnston (1) reported that urinary lactose excretion may be a good biomarker of lactation performance. The mean urinary lactose excretion of the 13 lactating women in their study was 50-fold higher than that of 15 nonlactating women, and the urinary lactose excretion of women who nursed more than three times daily was significantly higher than that of women who nursed less frequently. Because lactose is the most osmotically active component of human milk, lactose synthesis may influence milk volume (5). The concentration of lactose in human milk varies little from day to day or from feeding to feeding (6). The amount of lactose secreted into the plasma is approximately 1% to 2% of the amount in breast milk (2). It is feasible, therefore, that the concentration of lactose excreted in the urine, though small, could be proportional to the volume of milk secreted.

The purpose of this study was to validate urinary lactose excretion as a sensitive, specific biomarker of lactation performance by comparing urinary lactose excretion with breast milk output, estimated by the test-weighing procedure,
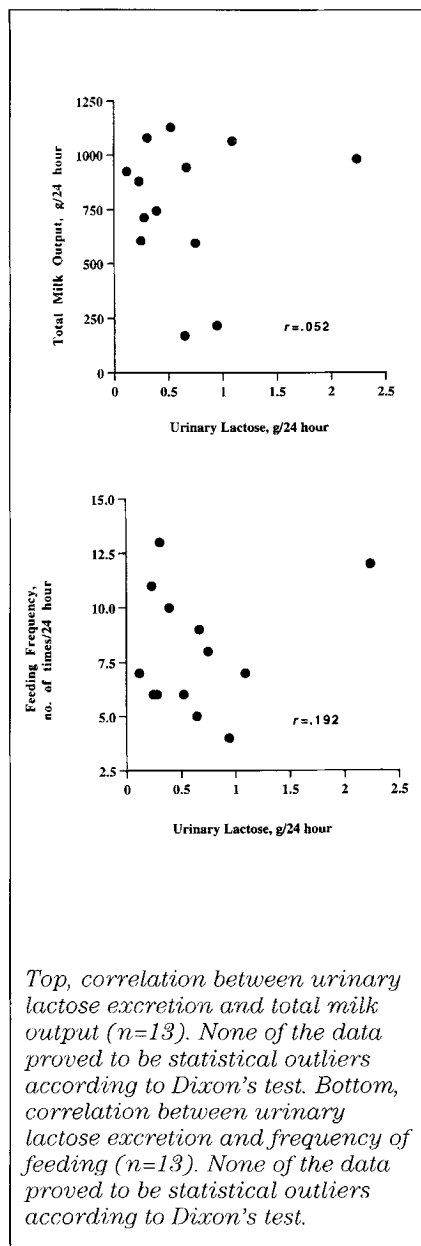
Table
Mean (±standard deviation) urinary lactose levels during a lactation and nonlactation time period in 13 women

Measure	Lactation	Nonlactation	P
Total urinary lactose excretion (g/24 h)	0.64±0.16	0.07±0.03	.005
Urinary lactose concentration (mmol/L)[a]	1.22±0.22	0.12±0.03	.001
Lactose:creatinine molar ratio[b]	65.5±14.5	5.9±2.0	.008

[a]To convert mmol/L urinary lactose to mg/L, multiply mmol/L by 342.3.
[b]To convert lactose:creatinine molar ratio to mg ratio, multiply molar ratio by 2.6.

Top, correlation between urinary lactose excretion and total milk output (n=13). None of the data proved to be statistical outliers according to Dixon's test. Bottom, correlation between urinary lactose excretion and frequency of feeding (n=13). None of the data proved to be statistical outliers according to Dixon's test.

in a group of women studied twice, during lactation and a nonlactation state.

METHODS
As part of a longitudinal study of calcium and zinc metabolism during pregnancy and lactation, 24-hour urine samples were collected from 13 healthy women in lactating and nonlactating states (7). The protocol for selection and treatment of the human subjects was approved by the Berkeley Committee for the Protection of Human Subjects. All subjects reviewed the study procedures and gave written consent.

All urine samples for the lactation period were collected between 7 and 9 weeks after delivery; the nonlactation samples were collected before pregnancy in 11 women, during the first trimester in 1 woman, and after weaning in another woman. The 24-hour urine samples were collected by the subjects in their homes. No preservative was used and the samples were not routinely refrigerated during the collection period. After collection, the samples were made into aliquots and stored at –20°C until analyzed to determine lactose and creatinine levels. To determine whether urinary lactose is susceptible to hydrolysis while stored at room temperature during the collection period, we measured the lactose concentration of a reference urine sample that was stored at room temperature for 0, 6, 12, 24, 36, or 48 hours. Length of storage had no effect on urinary lactose concentrations; the coefficient of variation for the samples at all six time points was 7.5%. We concluded that storage conditions during collection do not affect urinary lactose concentration.

Urinary lactose concentration was measured using a kit (Boehringer-Mannheim, Mannheim, Germany); urinary creatinine level was measured using the alkaline picric acid method. The coefficient of variation for repeated measurements of urinary lactose concentration was 4.1%.

Total output of breast milk was determined from the measurement of the

infant's weight on an electronic balance sensitive to 1 g (Sartorius, Model LC34, Dublin, Calif) before and after each feeding for a 72-hour period. Recorded infant weights were corrected for evaporative losses during the feeding period (8). The number of feedings during the test-weighing period were recorded by the mothers. Five of the women collected their urine samples during the time of the test-weighing procedure. The other eight subjects collected urine within 3.9±3.7 days of the test-weighing procedure.

Significant differences between urinary lactose excretion at the lactation and nonlactation time points were determined using Student's t test for paired data. Pearson correlation coefficients were determined for urinary lactose excretion and milk output.

RESULTS AND DISCUSSION
Eleven white and two Hispanic women participated in the study. Mean (±standard deviation) age of the women was 30±3 years and their prepregnant weight and body mass index (kg/m²) were, respectively, 61.3±6.8 kg and 22.5±2.6. Ten of the women were primiparas; the other three were multiparas.

Total urinary lactose excretion and urinary lactose concentration were significantly higher during lactation than during the nonlactation period ($P<.005$ and $P<.001$, respectively) (Table). Although total urinary lactose excretion was elevated, only a 10-fold difference was observed in this study compared with a 50-fold difference reported by Strand and Johnston (1). This variance may be related to the fact that we used 24-hour urine samples, whereas Strand and Johnston used only single-spot urine samples. The ratios of moles of lactose to moles of creatinine during lactation vs nonlactation were 65.5±14.5 vs 5.9±2.0 (mean±standard deviation) in our study. Strand and Johnston reported that the lactose to creatinine molar ratios were 60±11 during lactation vs 1.1±0.4 during nonlactation.

To be a valid biomarker of lactation performance, a high correlation between urinary lactose excretion and total milk output is required. As shown in the Figure, urinary lactose excretion was not correlated with total milk output or frequency of feeding in our subjects. We also found no correlation between milk output and lactose:creatinine molar ratio ($r=.155$) or lactose concentration ($r=.052$). When these same correlations were performed in the five women for whom the urine collection coincided with the test-weighing measurement, no significance was observed. Strand and

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RESEARCH AND PROFESSIONAL BRIEFS

Johnston (1) found that the urinary lactose:creatinine molar ratio was significantly lower in the four women who nursed less than three times per day vs those who nursed more than three times. Kerver and coworkers (9) reported a significant correlation between total 24-hour milk volume and urinary lactose concentration in a first morning void (*P*=.017). In agreement with our results, however, they did not find a correlation between milk volume and urinary lactose in 24-hour urine samples (*P*=.065). Expressed milk volume is considered to be a measure of total milk synthesis rather than total output. Possibly, milk synthesis occurs to a greater extent at night, so that the urinary lactose output in a first morning void reflects the amount of milk synthesis to a greater extent than lactose excretion in a 24-hour period.

APPLICATIONS

Lactation increases the urinary excretion of lactose 10-fold. However, neither total urinary lactose excretion or lactose:creatinine molar ratios are related to milk output or number of feedings in a day. Urinary lactose excretion cannot be used to predict milk volume or lactation performance.

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Management of intractable childhood seizures using the non–MCT oil ketogenic diet in 20 patients

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Two or more forms of the ketogenic diet are commonly used today, the medium-chain triglyceride (MCT) diet and the non-MCT high-fat diet, which demonstrate varied results in control of myoclonic seizures (1,2). In the MCT ketogenic diet, MCTs provide 50% to 70% of total energy (3,4), and although many patients tolerate an MCT diet, others must abandon it because of gastrointestinal intolerance (5,6). The success rate in controlling seizures with an MCT-based diet has been approximately 50% for persons between the ages of 2 and 5 years (4).

The purpose of this study was to determine whether the initiation of a non-MCT high-fat ketogenic diet would continue to reduce seizure frequency and medication use in pediatric subjects 2 weeks after they were discharged from the hospital. The success rate of eliminating or substantially decreasing seizure activity with a non-MCT diet has been 67% according to a study of 58 subjects (2).

METHODS

Twenty pediatric patients (aged 15 months to 11 years) with a history of poor control of epileptic seizures who were admitted to Miami Children's Hospital between January and May 1995 were recruited for this study. A non-MCT ketogenic diet with a 4:1 ratio of fat to nonfat was used. Approval by the

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Children's Hospital Institutional Review Board was not required for these subjects because the ketogenic diet was not considered an experimental treatment. At the time of admission, medical evaluation by the primary physician determined the length of fasting necessary for metabolic ketosis to occur in these subjects (usually 12 to 72 hours) and was dependent on age and blood glucose response. Our hypothesis was that seizure reduction would occur as a result of successful administration of a ketogenic diet.

The average hospital stay was 4 days. Patients were allowed unrestricted amounts of water before and during diet administration. In most cases, the diet was started at full strength when there was evidence of moderate to large urinary ketone spillage, as demonstrated by Ketostick (Bayer Corporation Division, Elkhart, Ind). The composition of the diets ranged from 3% to 5% carbohydrate, 7% to 13% protein, and 85% to 90% fat and was managed by a registered dietitian. Energy levels devised for patients ranged from 75% to 125% of the Recommended Dietary Allowances (7) as recommended by the literature (8) and by the patient's hunger (9). The Table illustrates an individualized patient meal plan, with weighted food portions.

Patients were discharged from the hospital when diet tolerance, blood glucose normalization, and seizure control were achieved. Patient urinary ketones levels ranged from +2 to +3 at the time of discharge. Dietary counseling occurred throughout hospitalization, and caregivers were given at least three sample menus to follow. Communication continued by telephone and in follow-up appointments.

RESULTS

During the 2-week period of consuming the diet, hypoglycemia was experienced